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Latex Induced Occupational Asthma in a Surgical Pathologist

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Running Heading: Latex Induced Occupational Asthma

Key words: Occupational Asthma, Latex Allergy, Formaldehyde, Xylene, Pathology, Health care worker.

Abbreviations:

ACD: Allergic contact dermatitis

FDA: Food and Drug Administration

FEV1: Forced expiratory volume in one second

FVC: Forced vital capacity

HMW: High molecular weight

ICD: Irritant contact dermatitis

IgE: Immunoglobulin E

NIOSH: National Institute for Occupational Safety and Health

NRL: Natural rubber latex

OA: Occupational asthma

OSHA: Occupational Safety and Health Administration

PAPR: Powered air- purifying respirator

PEFR: Peak expiratory flow rate

PPE: Personal protective equipment

RADS: Reactive airways dysfunction syndrome

RAST: Radioallergoimmunoabsorbent assay

Outline:

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Abstract

Context: Latex allergy and sensitization has been an important problem facing health care workers. Providing a latex safe environment is the intervention of choice.

Case Presentation: A 46-year-old surgical pathologist presented with increasing shortness of breath for the previous 4 years. Twenty years prior to presentation, he noted a pruritic, erythematous rash on his hands, associated with latex glove use. Fourteen years prior to presentation, during pathology residency, he developed a non-productive cough, wheezing and an urticarial rash, temporally associated with powdered latex glove use. These symptoms improved while away from work. At presentation, he had one-flight dyspnea. Skin prick test was positive for latex and pulmonary function testing showed mild obstruction, reversible with bronchodilator. As the patient was at risk for worsening pulmonary function and possible anaphylaxis with continued exposure, he was removed from the workplace as no reasonable accommodation was made for him at that time.

Discussion: His presentation is consistent with latex induced occupational asthma. Initially noting dermal manifestations, consistent with an allergic contact dermatitis secondary to accelerators present in latex gloves, he later developed urticaria, flushing and respiratory symptoms, consistent with a type I hypersensitivity reaction to latex. He also has reversible airways disease with significant improvement of peak expiratory flow rate and symptoms, when away from work.

Relevance to Clinical or Professional Practice: The ideal treatment for latex sensitization is removal from, and avoidance of exposure. Clinicians should consider occupational asthma when patients present with new onset asthma or asthmatic symptoms that worsen at work.

Case Presentation

A 46-year old, male, surgical pathologist presented to our clinic complaining of a four-year history of increasing shortness of breath. He had been in good health until 20 years prior while in medical school, when he noted a pruritic, erythematous rash on the dorsal aspect of his hands whenever he wore latex gloves. He often applied steroid cream to the rash, but it usually did not resolve unless he refrained from using latex gloves. This rash, associated with latex glove use, persisted during his internal medicine residency. Approximately 14 years before presentation, at the beginning of his pathology residency, he noted that the rash involved his arms. He developed an episodic, nonproductive cough, wheezing, and occasional chest tightness, which occurred at work with powdered latex glove use. These symptoms were mild and did not interfere with his vigorous exercise program. He did not seek medical attention.

After completing his residency, he worked as a hospital-based surgical pathologist. Typical daily activities involved cutting tissue, doing frozen sections and preparing slides. He changed gloves several times each day. He did reasonably well until 4 years prior to presentation (1993) when his symptoms worsened. He now experienced cough and dyspnea within 30 minutes of starting work. These symptoms, which continued throughout the workday and improved once he left work, seemed especially severe on the first day of the workweek and worsened as the week progressed. Xylene and formaldehyde exacerbated his symptoms. He noted an intermittent rash on his upper extremities and torso, occasional flushing with exposure to latex, post-nasal drip, progressive dyspnea on exertion, and dyspnea and coughing when he laughed. He noted heavy breathing if he "flipped" his gloves off and described an episode of "passing out" one year prior when he "flipped" his gloves off and placed his hands over his mouth and nose. He was taken to a local Emergency Room where he was diagnosed as having had a vasovagal episode. He was returned to work without intervention.

The patient's wife and co-workers started commenting on his cough, noting that that he "breathed heavily". He became self-conscious about his cough and about constantly having to clear his throat. There was no seasonal variation to his symptoms. The patient attempted to reduce his exposure to powdered NRL gloves, formaldehyde and xylene. For example, he switched to non-powdered latex gloves, although his co-workers continued to use the powdered form. He replaced eyecups on the microscope once he realized that they contained latex. He instructed his staff to allow an hour for drying slides fixed with formaldehyde and xylene, before sending them to him to be read. His symptoms persisted however, prompting him to seek medical attention.

The patient subsequently consulted with an allergist, an otorhinolaryngologist, and a dermatologist. Skin biopsy of his rash revealed changes consistent with acute urticaria. Latex skin prick tests were positive to latex glove extracts. Skin prick tests were positive to dust, cat, dander and mold antigens and a computerized tomography scan of the sinuses revealed nasal polyps in the maxillary sinus. He was diagnosed with chronic sinusitis, asthma and allergic rhinitis. Treatment included antibiotics and a steroid taper. He was started on Serevent ® (GlaxoSmithKline, Research Triangle Park), Flovent ® (GlaxoSmithKline, Research Triangle Park) and Proventil ® (Schering, Kenilworth) inhalers and returned to work with the recommendation that he use a surgical mask while at work. His symptoms continued to progress, and he presented to us 2 months later by which time he was experiencing single flight dyspnea.

His past medical history was remarkable for hypertension, nasal polyps, and near syncope. He denied any previous diagnosis of asthma, allergy, hives, or anaphylaxis. His family history was remarkable for asthma in a sister and paternal uncle. He denied alcohol, cigarette, or illegal drug use, as well as denied allergies to medications or environmental substances. He gave a history of chest tightness when he ate fruit such as banana, avocado and kiwi. His occupational history was remarkable for work in the medical field. (SeeTable 1) On physical examination, he was a pleasant, well-nourished, well-developed white male in no acute distress whose vital signs were within normal limits. His examination was remarkable for a body mass index of 30, hyperemic conjunctivae, boggy nasal mucosa, an erythematous urticarial rash on his right shoulder, and diffuse expiratory wheezing.

Laboratory evaluation revealed a normal electrocardiogram. Chest X-ray showed poor inspiration, computerized tomography of the chest showed mild bronchial wall thickening consistent with mild airways disease, pulmonary function testing was remarkable for mild obstruction with acute bronchodilator response, (see Table 2) and radioallergoimmunoabsorbent assay (RAST) test for latex IgE antibody was negative. His peak expiratory flow rate (PEFR) diary during an 11-day work-period, and a subsequent 6 day vacation-period showed significant improvement (20% in the morning; 22% in the evening) while he was away from work (Table 3), and progressive improvement during successive days of vacation (Figure 1).

The provision of a latex safe environment was explored with hospital administration and deemed not feasible at that time. A full-face dual cartridge respirator was recommended and trialed in consultation with a certified industrial hygienist. However, it interfered with the patient's ability to communicate and he was unable to tolerate wearing it for an 8-hour day. It was felt that he was at risk for potentially fatal anaphylaxis, as well as irreversible and impending structural damage to his lungs given his long history of exposure and disease severity. In order to eliminate exposure to NRL the patient was removed from the workplace. He was advised to avoid contact with latex, carry injectable epinephrine, and wear a medic alert bracelet. Despite removal from the workplace shortly after presentation, the patient's pulmonary status did not improve. He is maintained on steroids and immunosuppressive agents and has not been able to return to work as a surgical pathologist.

Discussion

Latex Allergy and Sensitization

The use of powdered high-protein natural rubber latex (NRL) gloves is recognized as the major environmental risk factor for latex sensitization and allergy in the healthcare field (Levy et al. 1999; Wild and Lopez 2003). The widespread use of NRL gloves in the health care industry started in the 1980's as health care facilities complied with the Universal Precautions (OSHA 1991). After the first report of a case of immediate hypersensitivity to NRL (Nutter 1979), NRL allergy became increasingly recognized as a problem among healthcare workers (HCWs) (Garabrant and Schweitzer 2002). NRL, used in the production of latex gloves, is derived from the milky sap of the commercial rubber tree - *Hevea Brasiliensi* (Atkins 1999). The sap of this tree is a complex mixture of protein, lipid and phospholipid. The protein content varies depending on country of harvest location, environmental conditions, and manufacturing process. Sixty of the 240 proteins in NRL have been found to be allergenic (Levy et al. 1999).

Freshly harvested latex is treated with ammonia and other preservatives in order to prevent its deterioration during transport to factories, and then with antioxidants and accelerators before being shaped into the desired object. Increased washing time in glove manufacture can lead to a decrease in the amount of soluble protein in the final product. (Yunginger, Jones et al.) hence decreasing the antigenicity of the glove. The product is frequently dry-lubricated with cornstarch or talc powder to improve ease of donning the glove. Latex allergen elutes onto the powder providing a source for respiratory exposure (Yunginger

Jones et al. 1994). Notably, synthetic rubber elastomers (butyl rubber, polymers of 2-chlorobutadiene, co-polymers of butadiene and acrylonitrile) do not cause or contribute to allergic sensitization; people who are sensitized to NRL proteins can safely use products made from synthetic rubbers (OSHA 1999; Renaud 1993).

The majority of reactions associated with NRL can be classified into three main categories. They are irritant contact dermatitis (ICD), allergic contact dermatitis (ACD) and an immediate hypersensitivity reaction (Felt-Ahmed et al. 2003). ICD is confined to the skin and occurs when the skin has direct contact with the glove. ICD represents a type of contact dermatitis and is not allergic in nature. The second type of reaction, ACD, is a delayed hypersensitivity reaction (type IV) felt to be a result of exposure to the accelerators, which can lead to the activation and release of lymphokines by sensitized T lymphocytes, rather than to the latex itself (Atkins 1999). Endotoxins, which may be present as contaminants, have also been implicated as causing ACD (Charous et al. 1997). Features of ACD are pruritic rash, local erythema, swelling, blistering, weeping and crusting. These symptoms generally occur 1 to 2 days after exposure, but also may occur from several hours to several days post exposure (Felt-Ahmed et al. 2003).

The third type of reaction, the type I, immediate type hypersensitivity reaction, which relies on previous sensitization of the immune system to latex antigens and to the generation of IgE antibodies directed specifically at latex proteins, is the most serious of the three (Atkins 1999; Vandenplas et al.1995). Signs and symptoms include asthma, rhinitis, conjunctivitis, generalized urticaria and mucous membrane swelling. Anaphylaxis, the most dreaded complication, may also occur in a sensitized patient, and has been recorded to have occurred as a result of donning gloves, being in the presence of others who have put on gloves, during surgery, and during dental and medical examinations (Vandenplas et al. 1995). In 1991 a latex barium enema tip associated with 16 deaths was recalled by the FDA. This led to an increased awareness of the risk of life threatening type 1 allergy associated with NRL devices (Gelfand 1991). Sensitization occurs after multiple exposures over a highly variable time, the latency period ranging from several weeks to as long as 30 years (Malo et al. 1992). Once sensitization occurs, there is considerable variability in the type and severity of allergic symptoms, occurring within 30 minutes (anaphylaxis, angioedema), to over hours and days after exposure. Asthma symptoms are highly variable in their onset, duration and intensity, the more severe cases being associated with multiple and prolonged exposures occurring over many months to years (Felt-Ahmed et al. 2003).

The prevalence of latex sensitization has been estimated to be between 5% and 17% in HCWs (Malo et al. 1992*),* as opposed to being between 5% and 10% in the general population (Felt-Ahmed et al. 2003). Some of the factors associated with an increase in the risk of latex sensitization among HCWs are the duration of exposure and the intensity of exposure to NRL gloves. Intensity of exposure is measured by the number of pairs of gloves used per day, and the amount of powdered glove use (Garabrant and Schweitzer 2002). The mechanical and irritant reaction to the powder may lead to a breakdown of the skin barrier further enhancing exposure to the latex protein (Levy et al. 1999). In addition, the powder disseminates into the environment carrying the latex protein with it, providing a respiratory route of exposure (Baur et al. 1993). An increase in latex sensitization is seen with particular jobs and departments in healthcare probably as a result of a relatively higher exposure to NRL gloves. Laboratory workers have been found to have the highest incidence of latex sensitization, 4% per year, whereas the incidence of latex sensitization among HCWs in general has been estimated at 1% to 2.5% per year; pathology staff has been found to have a 14% prevalence of latex sensitization (Garabrant and Schweitzer 2002).

Atopic individuals are more easily sensitized to allergens and as such, are at greater risk of developing a latex allergy than individuals who are not atopic (Felt-Ahmed et al. 2003). Atopy is a hypersensitivity state or allergy with hereditary predisposition. Atopic individuals may have a personal or family history of eczema, asthma or hay fever, or a tendency to develop specific IgE antibodies after exposure to common environmental substances, although many do not. The tendency to develop some form of allergy is inherited, but the specific clinical form such as hay fever, asthma, or eczema, is not *(*Wild and Lopez 2003). Skin tests to common environmental allergens such as pollen, animal dander, molds, and house dust mites are used to evaluate atopic status. One looks for the immediate IgE mediated wheal and flare reaction. Clinical associations have been reported between latex allergy and allergy to several fruits and vegetables, such as avocado, kiwi fruit, banana, potato, tomato, chestnut and papaya (Beezhold et al. 1996). Several latex allergens (such as Heb b2, 5, 6.02, and 7) have varying degrees of amino acid sequence homology with allergens in seed producing plants (Wagner and Breiteneder 2002). Some patients report that food allergy preceded the latex allergy and others report the converse (Beezhold 1996).

Sensitization can be documented by the use of a skin prick test using extracts prepared from suspected substances in the work environment, such as latex. Detection of specific IgE antibodies suggests a cause and effect relationship. Licensed extracts of latex for skin testing, available in Europe, have been found to be safe and reliable for detecting latex-specific IgE. The United States does not have licensed commercial latex extracts. As a result, skin testing is done with unstandardized office-prepared latex extracts, which vary widely in allergen content (Ownby 2003). Specific IgE antibodies can also be studied in vitro using a blood test, the radioallergoimmunoabsorbent assay (RAST) (Wild and Lopez 2003). Tests for latex-specific IgE such as the RAST are less sensitive and specific than the skin prick tests, with sensitivity ranging between 73% and 80%, and specificity ranging between 90% and 97% (Ownby 2003).

Latex Induced Occupational Asthma

Occupational asthma (OA) can be defined as the presence of variable airflow obstruction and bronchial hyper-responsiveness caused by a substance found in the workplace (Tilles and Jerath-Tatum 2003). OA differs from preexisting asthma, which is exacerbated by exposure to agents in the workplace (Wild and Lopez 2003). However, OA may occur in conjunction with pre-existing asthma, as OA involves the new onset of sensitization to a workplace antigen or allergen with the development of respiratory disease. A person with pre-existing asthma and allergies may develop OA to a workplace allergen. Another feature of OA is the occurrence of nasal, ocular or contact urticarial symptoms that precede asthma symptoms. The presence of these symptoms is helpful, but not necessary, in establishing the diagnosis.

Other features include the association of prolonged exposure with worsening asthma symptoms at work, the development of more pervasive symptoms while at work, and the presence of a latency period between the initial exposures to the inciting agent where symptoms may develop from weeks to more than 20 years after exposure (Chan-Yeung 1987; Tilles and Jerath-Tatum 2003; Wild and Lopez 2003). Reactive airways dysfunction syndrome (RADS) is a form of OA that does not require a latency period. RADS can occur acutely, within 24 hours, after one single exposure to an irritant (Tilles and Jerath-Tatum 2003). OA symptoms may resolve in some individuals while others remain symptomatic for years. Approximately 10% of adult asthma cases are attributed to an occupational etiology (Blanc and Toren 1999). More than 250 agents encountered in the workplace have been shown to induce asthma in susceptible individuals (Wild and Lopez 2003).

Atopic individuals are at greater risk of developing OA, especially when working in an industry where high molecular (HMW) proteins such as latex proteins are present. Other HMW proteins known to cause OA are flour and animal antigens (Wild and Lopez 2003). Allergic OA is seen in individuals who develop sensitization to a specific chemical agent in the workplace. Persons with allergic OA tend to develop bronchospasm and airway inflammation upon exposure even to low concentrations of the specific workplace agent to which they are sensitized (Paggiaro et al. 1994). NRL induced occupational asthma, an IgE -mediated process, is initiated when the allergen-bearing particles deposit unto the mucosal surfaces of the respiratory tract. Of the HCWs estimated to be sensitized to latex, 41-69% of them are estimated to have respiratory symptoms with exposure (Lagier et al. 1992).

Various criteria are used in making the diagnosis of OA. A significant post-bronchodilator response is considered to have occurred if pulmonary function tests (PFTs) demonstrate an increase in FVC or FEV1 of 12% above baseline and an absolute change of 0.2L (American Thoracic Society 1991). Methacholine challenge testing, the gold standard for establishing the diagnosis of asthma, can also be used to show nonspecific bronchial hyperreactivity. An abnormal test result is defined by the concentration of methacholine that drops the baseline FEV1 by 20% (Tan and Spector 2003). Medical and work histories may be used to help ascertain a temporal association between the patient's symptoms and work, as well as to rule out other causes for the symptoms.

One recommendation for confirming the diagnosis of occupational asthma, using pre and post-shift spirometry or PEFR, is by showing a significantly decreased obstructive pattern at work as compared to being away from work. For example, PEFR should be measured approximately every 2-3 hours during a two-week period at work, and during a 1-2 week period away from work. OA is confirmed by finding a 20% or greater reduction in PEFR at work versus away from work, or by finding at least a 20% diurnal variability of mean work PEFR, with the disappearance of this variability when away from work (Tilles and Jereth-Tatum 2003). PFTs are most useful in suggesting an occupational cause for asthma when they show a decrease in forced expiratory volume in one second (FEV1) of at least 15% when comparing results obtained before and after a period of work (Greaves 2003). The diagnosis of occupational asthma is usually confirmed by a combination of findings. The history and physical exam should be consistent with this diagnosis, spirometry or methacholine challenge testing should demonstrate variable airflow obstruction, and serial peak flows should confirm that bronchial hyperreactivity is triggered by workplace exposures to specific agents.

Role of Formaldehyde and Xylene

Formaldehyde is an upper respiratory tract irritant, exacerbating bronchial airflow obstruction or hyperreactivity. It can exacerbate asthma and precipitate wheezing in those with underlying asthma or bronchial hyperreactivity. Formaldehyde may cause an immune response by forming a hapten, a complex of a protein and a low molecular weight compound, which can induce an IgE response, although this is uncommon (Rutchik 1999). Xylene, an aromatic hydrocarbon used in medical technology as a solvent and fixative, may exacerbate asthma and rhinitis. Other agents to which the patient may have been exposed during his daily work as a pathologist not identified by him as being specific triggers to his symptoms, but that are associated with respiratory and dermatological symptoms, are glutaraldehyde, phenol and ethylene glycol (Rutchik 1999).

Treatment and Workplace Accommodation

Disability from occupationally induced allergies is compensable under workers' compensation law (Philips et al. 1999). A worker with OA or NRL-induced anaphylaxis is considered to be 100% impaired from performing his or her specific job if the job entails exposure to the causative agent (American Thoracic Society 1993; Bernstein 2002). Under the Americans with Disabilities Act (1990), reasonable workplace accommodation must be made to allow a disabled worker to perform the "essential functions" of the job. The ideal treatment for latex sensitization is prevention of exposure, best achieved by identifying and removing all latex-containing products in the workplace. Latex aeroallergen levels are significantly reduced when medical centers eliminate powdered NRL gloves from the work environment, replacing them with non-powdered synthetic rubber gloves (Swanson et al. 1994). This workplace modification has been found to be most effective, and associated with an improvement in respiratory and dermatological symptoms in HCWs, as well as in a reduction in the number of new cases of latex sensitization and allergy (Bernstein 2003; Hunt 2002; Saary 2002; Swanson 1994). It has also been shown to be cost effective, considering the cost incurred by disability from latex allergy and asthma (Allmers 2002; Philips 1999)

Many medical devices and products, as well as many common household and everyday items contain NRL. However, identifying latex containing products was made simpler as of 1998 when the Food and Drug Administration (FDA) mandated that all NRL-containing medical devices be labeled as such, and that healthcare sites should provide non-latex containing alternatives. The FDA concluded that this intervention is affordable for manufacturers (U.S. FDA). Extensive lists of NRL containing products and latex-safe alternatives are also available (Spina Bifida Association of America 2004). Despite this however, it is difficult to render and maintain an environment completely latex-free. Furthermore, NRL containing items may also be inadvertently brought into an area. As a result, latex-safe is the preferred term.

Prevention of exposure may also be carried out through engineering and industrial hygiene controls, and through the use of personal protective equipment (PPE). Latex aeroallergen levels may be monitored, and engineering controls can include exhaust ventilation equipment (Reiter 2002), although the use of a laminar flow glove changing station has not been shown to reduce latex aeroallergens (Swanson 1994). Work practice controls, such as cleaning the area might help to eliminate or minimize the hazard. Environmental controls such as HEPA-filtered vacuuming and wet wiping of surfaces with isopropyl alcohol may reduce latex allergen on surfaces (Reiter 2002).

The worker may also use PPE, such as a respirator. Respirators can provide additional protection and mitigate the hazard but are not the method of choice for controlling exposures. There are various categories of respirators. Air- purifying respirators may use negative pressure (the user pulls air through the respirator) or air is supplied through a powered source (powered air- purifying respirator or PAPR). They remove much of the toxicant from the inhaled air by filtration, adsorption or absorption. Atmosphere-supplying respirators, such as the self-contained breathing apparatus (supplies air from a source such as a tank carried by the user), and the airline respirator (uses air supplied via a hose from a distant source), provide air from an independent source as opposed to purifying ambient air.

Most respirators require a tight seal between the mask and the user’s face although some are loose fitting. Masks are quarter, half or full face depending on the portion of the face that is covered (Harber et al. 2005; NIOSH 2005). Laminar flow HEPA-filtered helmets have been found to be effective in reducing the symptoms of latex-induced asthma, rhinitis and conjunctivitis (Laoprasert 1998). Respirators may interfere with vision, hearing, mobility, ability to communicate, and with the use of tools such as stethoscopes and microscopes. They may be uncomfortably warm with tight fitting head straps and may also lead to increases in resistance to breathing, dead space, and physical load. These factors, among others, may contribute to a functional inability to keep the respirator on for more than a brief period of time in some persons. Recommendations of a certified industrial hygienist should be used when available (Harber et al. 2005; NIOSH 2005).

Sensitized workers with severe asthma, and other life-threatening allergic reactions must be removed from the workplace if exposure cannot be prevented, as the asthmatic response can occur at minute levels of exposure (Ehrlich 1994). Although not documented in individuals with OA due to NRL, evidence from other sensitizing agents such as western red cedar asthma and toluene diisocyanate indicates that repeated exposures to the inciting agent can increase the severity of the asthma and the disease process may even progress after removal from exposure (Butcher et al. 1982; Bnks 1980; Chan-Yeung 1982; Cote and Chan-Yeung 1990). Ultimately, irreversible lung damage and death can result from repeated exposure (Banks et al. 1990; Chan-Yeung 1987).

Removing the employee from the workplace has personal, social and economic implications. The latex-allergic HCW may experience psychological distress secondary to coping with the adjustment and may respond with anger, depression, anxiety and denial. Self-esteem, interpersonal relationships and economic well-being may be adversely affected when an individual is unable to maintain his current profession with the possible loss of future earnings or forced early retirement. These factors, among others, may lead the HCW to delay seeking much needed medical attention (Charous, Blanco, et al. 2002). In addition to eliminating exposure to latex, the treatment for OA is same as for other types of asthma (Wild and Lopez, 2003). Workers with latex sensitization and latex induced OA should be counseled to wear a medic alert bracelet and carry injectable epinephrine with them at all times. They should also be counseled as to what items contain latex and to avoid dermal, mucosal or serosal contact with them (Howarth 2001).

Conclusion

This case illustrates that of a surgical pathologist whose presentation is consistent with a diagnosis of latex induced occupational asthma. It shows how exposure to a HMW protein, latex, led to an allergic contact dermatitis. Repeated exposure to the inciting agent over a latency period of several years led to latex sensitization and ultimately to latex induced occupational asthma in this atopic individual. He does not give a clear history of anaphylaxis, but was diagnosed with “near syncope”of unknown etiology after flipping his gloves off and placing his hand over his nose and mouth, after which he was returned to work without intervention. Skin prick test, which is diagnostic for the presence of IgE mediated allergy to latex, was positive to several latex-containing extracts. Although his serum IgE or RAST, to one type of latex protein, was negative, the laboratory to which it was sent reports a 30% false negative rate (Hamilton 1999). The patient's medical and occupational history, in combination with his spirometry and PEFR measurements, support the diagnosis of occupational asthma, reversible airways disease responding to bronchodilators with symptoms that are worse at work and improve away from work. Formaldehyde and xylene probably acted as irritants, exacerbating his pulmonary symptoms.

The mainstay of treatment for latex-induced occupational asthma is to prevent contact of the worker with the inciting agents. Creating a latex-safe environment is the provision of choice (Charous, Tarlo al. 2002). However, this provision was not made at the time. Given the long period of the patient's exposure and the severity of his disease, there was concern that his pulmonary function would continue to decline with continued exposure, and that he was at risk for anaphylaxis. Removal from the workplace was felt to be the best way to protect the patient from exposure. Despite removal from inciting agents, the patient's symptoms and pulmonary status did not improve. He remains out of work, and is maintained on steroids and immunosuppressive agents. Had his condition been identified and removal from exposure occurred sooner, his disease may not have progressed. Prompt identification of latex allergy and sensitization, as well as reduction or elimination of the hazard, may allow the HCW to continue working in his environment and prevent progression of disease. Clinicians should consider occupational asthma in patients who present with new-onset asthma, or who present with asthma symptoms that worsen during or after work.

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Table 1: The chronological relationship between the patient's occupational exposure and appearance of symptoms.

Date	Occupation	Symptoms
1977	Medical Student	Rash on dorsum of hands with latex glove us, does not clear with steroid use
1979	Internal Medicine Resident	Rash on dorsum of hands with latex glove use continues
1984	Pathology Resident	Rash on hands and arms, urticaria, wheezing, chest tightness, chronic cough
1987	Pathology Attending	Diagnosed with nasal polyps
1993	Pathology Attending	Notes dyspnea within 30 minutes of work and with coughing and laughing
1996	Pathology Attending	Allergist evaluation results in diagnosis of asthma and allergic rhinitis; Emergency Department evaluation results in diagnosis of "near syncope" after flipped off gloves and covered mouth and nose with hands
1997	Pathology Attending	Single flight dyspnea, presents to our clinic; no reasonable accommodation made at work, removal from the workplace

Table 2: Spirometry results before and after bronchodilator use showing forced expiratory volume in one second (FEV1)and forced vital capacity (FVC).

	Pre-Bronchodilator	% Predicted		Post-Bronchodilator	% Predicted		% change
FEV1 (Liters)	2.65	67%		2.98	75%		13%
FVC (Liters)	3.96	81%		4.47	91%		13%
FEV1/FVC	67%			67%			

Table 3: Mean AM and PM peak expiratory flow rates while at work and during vacation, measured in the AM prior to asthma medication and measured in the PM at bedtime prior to asthma medication.

Time	Mean PEFR – Work	Mean PEFR - Vacation	% Increase
AM	368 L/minute	443 L/Minute	20%
PM	361 L/minute	441 L/minute	22 %
% Increase	-2 %	-0.5%	
Range peak flows	320 – 425 L/min	340-550	

Figure 1: AM and PM Peak Flows recorded in 1997 on 11 consecutive days while at work (Sun, Nov. 2 – Wed, Nov 12) and on 6 consecutive days while on vacation (Wed, Nov. 13 – Tues, Nov. 18).